COMPONENT PART NOTICE

THIS PAPER IS A COMPONENT PART OF THE FOLLOWING COMPILATION REPORT:

TITLE: Proceedings of a Workshop on Radiofrequency Radiation Bioeffects Held at Wachtberg-Werthhoven (Germany, F.R.) on 11-13 September 1984.

TO ORDER THE COMPLETE COMPILATION REPORT, USE AD-A157 090.

THE COMPONENT PART IS PROVIDED HERE TO ALLOW USERS ACCESS TO INDIVIDUALLY AUTHORED SECTIONS OF PROCEEDING, ANNALS, SYMPOSIA, ETC. HOWEVER, THE COMPONENT SHOULD BE CONSIDERED WITHIN THE CONTEXT OF THE OVERALL COMPILATION REPORT AND NOT AS A STAND-ALONE TECHNICAL REPORT.

THE FOLLOWING COMPONENT PART NUMBERS COMPRISE THE COMPILATION REPORT:

AD#: P004 959 thru P004 971 AD#:

AD#: AD#:

AD#: AD#:

Accession For
NTIS GRA&I
DTIC TAB
Unannounced
Justification

By
Distribution/
Availability Codes
Dist | Avail and/or Special

AD-P004 961

CRITICAL REVIEW OF SELECTED TOPICS ON BIOLOGICAL EFFECTS OF RADIOFREQUENCY RADIATION

Jerome H. Krupp
U.S. Air Force School of Aerospace Medicine
Brooks Air Force Base, TX 78235-5301, USA

Louis N. Heynick
Staff Scientist, SRI International
Menlo Park, California

Peter Polson
Consultant to SRI International
Menlo Park, California

INTRODUCTION

This report analyzes

Under the sponsorship of the U.S. Air Force School of Aerospace Medicine (USAFSAM), Brooks Air Force Base, Texas, SRI International is analyzing selected articles on the biological effects of electromagnetic radiation. To date, four reports embodying detailed critical reviews of 160 published papers have been issued: USAFSAM-TR-81-24 (November 1981), USAFSAM-TR-82-16 (May 1982), USAFSAM-TR-84-6 (March 1984), and USAFSAM-TR-84-17 (May 1984). The first two reports are available from the National Technical Information Service (NTIS), and the later two from NTIS or USAFSAM.

For convenience, RFR is used as a generic term to include all other designations in the literature for electromagnetic fields at frequencies up to 300 GHz. However, analysis of papers on the effects associated with the high electric and magnetic fields produced by high-voltage power-transmission lines is outside the scope of this paper. This paper

Some of the analyses in those reports served as the basis for a general review of RFR bioeffects, USAFSAM-TR-83-1 (March 1983), which is now undergoing updating; and some of the information presented in this paper was derived therefrom. In the time allotted, however, we will be able to discuss only a few major topics, which were selected as being of most significance with regard to possible hazards of RFR to the public at large. The papers under each topic are discussed in rough chronological order. These topics include:

SELECTED RFR-BIOEFFECTS TOPICS

EPIDEMIOLOGY

Cont'd pg. 61

Epidemiology as used here refers to studies of whether one or more health-related conditions can be associated statistically with purported or actual exposure of humans to RFR (in contrast with assessments based on extrapolation from data on animals to humans). Epidemiologic results tend to be based on imprecise estimates of exposure characteristics (frequency, power density, and duration). The extent to which the control group matches the exposed group is sometimes open to question. Because matching of all relevant factors except exposure is the basis for concluding that any observed differences between groups are related to the RFR exposure, selection of an appropriate control group is critical. Despite these limitations, such studies do provide almost the only information available on possible effects of actual RFR exposure in humans.

One of the early concerns was ocular damage from RFR exposure. Various cases of individuals with cataracts ascribed to RFR exposure have been reported from time to time. Indeed, it is likely that in some of these cases, occupational exposure to high levels of RFR had resulted in frank thermal damage to the eye. Not clear, however, was whether chronic exposures to low levels of RFR could be cataractogenic. The following epidemiologic studies of this question are representative.

Zaret et al. (1961) looked for eye defects in a group of 475 persons who were believed to have been exposed to RFR at 11 military and nonmilitary establishments; a group of 359 persons served as controls. The investigators

found a slight but statistically significant difference in defect scores between the two groups, but they expressed some doubt regarding the full validity of the scoring method used.

Cleary et al. (1965) examined Veterans Administration Hospital records of 2,946 Army and Air Force veterans of World War II and the Korean War who had been treated for cataracts. They selected a control sample of 2,164 veterans and, on the basis of military occupational specialties, classified each individual as a radar worker, a nonradar worker, or one whose specialty could not be discerned. In the radar group, they found 19 individuals with cataracts and 2,625 individuals without; in the nonradar group, 21 individuals with cataracts and 1,935 without. (The remaining 510 subjects were in the unspecified occupational category.) These differences between the radar and nonradar groups were not statistically significant.

Cleary and Pasternak (1966) reported on statistical analysis of the records of 736 microwave workers and 559 controls for minor lens changes, using a scoring range from 0 to 3. They reported that the defect scores increased with age for persons in both groups, but that the average score for the microwave group was significantly higher than for the control group. They suggested that this finding is an indication that exposure to RFR may have an aging effect on the lens; however, no cataracts or decreases in visual acuity were found.

Appleton (1973) reported on a study that covered 5 years. Military personnel identified as having been occupationally exposed to RFR from radar and communications systems were matched as closely as possible in age and sex with other military personnel on the same bases who had not been occupationally exposed. Several ophthalmologists independently examined exposed and control personnel (without knowing to which group each individual belonged) for opacities, vacuoles, and posterior subcapsular iridescence, taken as diagnostic precursors of cataracts. Each precursor was scored as either present or absent in each individual, and the binary data were used for statistical analyses by age group and number of persons per age group. The results indicated that more people in older age groups exhibited these precursors, but the pooled data from several installations showed no statistically significant differences between exposed and control groups.

As in other epidemiologic studies, detailed exposure histories (e.g., frequencies, intensities, durations) could not be determined with accuracy, if at all, for either the exposed or the control groups in these ocular studies. The exposed groups, however, likely did receive more RFR exposure. The relatively small research effort devoted to RFR ocular effects indicates that interest has waned in recent years.

Sigler et al. (1965) studied the occurrence of Down's syndrome (mongolism) in U.S. children and found an apparent correlation between this inherited condition and RFR exposure of the fathers of affected children before their conception. However, in a later study by Cohen et al. (1977) in which the original study of 216 children was expanded to 344 children with mongolism, each matched with a normal child of the same sex born at about the same time and whose mother was about the same age, no such correlation was found. Thus the earlier conclusion, based on a smaller sample, that exposure to RFR contributed to mongolism in offspring was not confirmed. No quantitative assessment of the extent of the fathers' exposures was possible.

The incidences of fetal anomalies and death rates reported in birth records for white children born in the vicinity of the Army Aviation Center at Fort Rucker, Alabama, between 1969 and 1972 were evaluated in a report by Peacock et al. (1971) and later by Burdeshaw and Schaffer (1977). Fort Rucker was of interest because of the concentration of radar units on or near the base. Taken together, these reports identified unusually high incidences of certain fetal anomalies and high fetal death rates in the two counties adjacent to Fort Rucker as compared with the corresponding statewide Alabama statistics, and at the Lyster General Hospital (Fort Rucker) as compared with other military and civilian hospitals. (A high incidence of fetal death at the Eglin AFB Hospital was also reported, but no further mention was made of the Eglin data in the remainder of the report.) There was also evidence, however, that the high rates for Fort Rucker could not be attributed specifically to the unquantified radar exposures at or near Fort Rucker on the basis of the birth record data: Coffee and Dale counties ranked only sixth and eighth for anomaly incidence among the 67 Alabama counties: Lyster Hospital's anomaly and fetal death rates were not significantly higher than several other comparable "nonradar" hospitals in Alabama and were in the range of values predicted from carefully controlled studies done in other states. The residences of mothers bearing anomalous infants were not clustered near radar sites, but many of the anomalies reported at Lyster occurred over a small time period, indicating a high anomaly-reporting rate for one or two physicians on the Lyster staff.

Pazderova (1971) published a report on the results of a battery of medical evaluations on 58 employees of Czechoslovakian television-transmitter stations. Exposure frequencies were estimated to range from 48.5 to 230 MHz at field intensities equivalent to 0-0.022 mW/cm^2, with a mean exposure duration of 7.2 years (10.6 h/workday). EKGs, heart and lung X-rays, standard blood tests, urinalyses, and liver-function tests were made, as well as ophthalmologic, neurologic, gynecologic, psychiatric, and psychological examinations. The only statistically significant finding was that the mean plasma protein levels were higher than "normal" values taken from the literature, a finding that the author described as unexplainable. The appropriateness of using literature control values is highly questionable.

In a later study, Pazderova et al. (1974) reexamined the effects of RFR on blood protein levels. The authors indicated that the only difference between exposed and control groups was that the members of the exposed groups had worked irregular shifts, whereas more than half of the control group had worked only morning shifts. The results for both groups showed that the individual levels of blood proteins and their fractions were within normal physiologic limits, but statistically significant differences were found between mean values for the exposed and control groups.

In our opinion, the absences in either study of a control group that had received virtually no RFR exposure renders questionable an interpretation that any differences found were due to RFR exposure. The altered values of blood proteins (which were within normal limits) were likely caused by other factors.

Klimkova-Deutschova (1974) surveyed various industrial worker populations in Czechoslovakia and assessed the health of workers exposed to RFR at 1-150 MHz, 300-800 MHz, or 3-30 GHz, with power densities, where specified, of 0.2

to 3.3 mW/cm^2, depending on their particular occupations. Changes were reported in brain-wave patterns and in blood sugar, proteins, and cholesterol levels, as compared with those in administrative (nonexposed) personnel. Numerical results, however, were not reported and statistical methods were not described.

Siekierzynski (1974) compared the health status and fitness for work of 507 persons in Poland occupationally exposed to pulsed RFR exceeding 0.2 mW/cm^2 average power density (other RFR characteristics not specified) with a group of 334 workers at the same installations exposed to less than 0.2 mW/cm^2. Clinical tests included ophthalmoscopic and neurologic examinations, supplemented by psychological tests and EEGs. No statistically significant differences between the two groups were found. In our opinion the lack of more definite RFR exposure data vitiates, but does not invalidate, the negative findings of this study; i.e., the results provide no evidence for RFR-induced effects on the health status of either group.

Kalyada et al. (1974) reported that their clinical examinations in the USSR of a group of specialists working with RFR generators in the 40- to 200-MHz range for 1 to 9 years showed occurrences of functional changes in the central nervous system, described as vegetative dysfunction accompanied by neurasthenic symptoms. No organic lesions were found, but among the many specific changes reported were deviations in the physiochemical and functional properties of erythrocytes and leukocytes. The authors also conducted experiments with human volunteers and reported functional changes in the thermoregulatory and hemodynamic systems and in the thermal, optical, and auditory "analysers." No RFR intensity values were given, however, for either the specialists or the volunteers; most of the findings were presented in narrative form, with no actual data; and the nature of the control group studied was not described. Consequently, this paper provides little basis for affirming or denying the occurrence of possible adverse effects of occupational exposure to RFR.

Sadchikova (1974) presented clinical observations on the health status of two groups of USSR RFR workers. The 1,000 workers in the first group were exposed to up to a few mW/cm^2, whereas the 180 workers in the second group were exposed to values rarely exceeding several hundredths of a mW/cm^2, both at unspecified "microwave" frequencies. A group of 200 people of comparable backgrounds but presumably not exposed to RFR served as controls. Sixteen kinds of symptoms were reported, including fatigue, irritability, sleepiness, partial loss of memory, lower heart-beat rates, hypertension, hypotension, cardiac pain, and systolic murmur. In the higher power density group, the indices for 5 of the 16 symptoms were higher than those in the lower power density group; they were lower for 9 symptoms and about the same for the remaining 2. Incidences in the control group were lower than those in either exposed group for 15 of the 16 symptoms.

Robinette and Silverman (1977) and Silverman (1979), in an attempt to establish whether exposure to RFR was associated with causes of death or with life expectancies, compiled the mortality records of personnel who had served in the U.S. Navy during the Korean War. By 1977 the records of about 20,000 deceased veterans whose military occupational titles indicated more probable exposure to RFR had been compared with the records of an approximately equal

number of less-exposed veterans. No quantitative exposure data were available. Although no differences between groups emerged in overall mortality rates or in the rates for about 20 specific categories of cause of death, death rates differed significantly for two categories: rates from arteriosclerotic heart disease were lower and those from trauma were higher in the RFR-exposed group. The trauma category included military aircraft accidents; and since a higher proportion of the exposed group had become fliers, attributing the higher trauma death rate to greater previous RFR exposure appeared unreasonable. Overall death rates for both groups were lower than for the general U.S. population of the same age.

The U.S. Embassy in Moscow was subjected to RFR exposure from 1953 until February 1977 (Pollack, 1979). Within rooms having the highest RFR levels (those with windows or doors in outside walls toward the irradiation sources), the average power densities were typically about 0.004 mW/cm^2 within 60 cm of a door or window and 0.0025 mW/cm^2 elsewhere in the room. The highest power density reported was 0.024 mW/cm^2, which occurred in one room during a 2-h period of unusual signal strength on 24 January 1976 (NTIA, 1981). Lilienfeld et al. (1978) compared the health of U.S. personnel assigned to the Moscow embassy from 1953 to 1976 with the health of those assigned to other U.S. Eastern European embassies. The investigators noted several limitations of their study but were able to conclude that no differences were discerned between the Moscow and control groups in total mortality or mortality from specific causes, nor between dependent children or adults of the Moscow and control groups.

Bielski et al. (1980) studied the effects of chronic occupational exposure to RFR in Poland. Two groups were studied: 88 persons in the radio and TV industry exposed 1-20 years to microwaves in the range from 3 to 7 GHz at 0.01-0.2 mW/cm^2 for an estimated 170 h per month, and 68 persons in the furniture industry exposed 1-16 years to HF RFR in the range from 7 to 30 MHz at approximately 0.2-10 mW/cm^2. Groups of 39 and 41 suitably chosen persons were used as unexposed controls for the microwave and HF groups respectively. The results showed that most of the people in the exposed groups complained of nonspecific symptoms--headaches, excessive irritability, increased perspiration, etc.--collectively called "typical symptoms of vegetative neurosis" by the investigators. The frequency of anomalous EEG records and the intensity of EEG changes were reported to be markedly higher among the HF than the microwave workers; however, no statistical tests on the data were reported. Also, it is not clear if the reported differences between exposed and control groups were RFR related or due to other factors. For example, the subjects from the furniture industry were involved with wood-gluing equipment that emitted HF RFR, but the workers were likely also exposed to fumes and vapors from the solvents in the glues.

Lester and Moore (1982a) claimed that U.S. counties with an Air Force base (AFB) operational during 1950-1969 showed statistically significant higher incidences of cancer mortality for that period when compared with counties without an AFB. We reviewed this study in detail and found that the data base used by Lester and Moore was incorrectly assembled. When we reassembled the data base correctly and analyzed it, we found that incidences of cancer mortality in counties with an AFB were not significantly different from incidences in population-matched counties without an AFB, for either males or females.

Lester and Moore (1982b) also reported that a neighborhood pattern of cancer incidence was found in the city of Wichita, Kansas, with the suggestion that it was related to chronic exposure of the general population to microwave radiation from airport radar systems. In their paper they ignored RFR exposure from other sources and used a theoretical model for exposure to airport radars that ignored inverse-square-law attenuation with distance, shielding effects of buildings, etc. The positive correlation of cancer incidence with "exposure" is therefore likely to be spurious. Although the relationship claimed by the authors between radar exposure and cancer incidence may exist, it was not demonstrated by the data and analysis presented in their paper.

In summary, none of these U.S., Polish, and Czechoslovakian epidemiologic studies offers clear evidence of detrimental effects associated with exposure of the general population or of selected occupational groups to RFR. The Soviet findings, which are consistent with the voluminous early Soviet literature, suggest that occupational exposure to RFR at average power densities less than 1 mW/cm^2 does result in various symptoms, particularly those associated with disorders of the central nervous system (CNS). Because the USSR symptomatology has not been reported in western studies and because of the marked differences between Soviet and Western publications in the procedures used for reporting data, it is difficult to accept the USSR epidemiologic studies at face value.

Cont'd

MUTAGENESIS, CARCINOGENESIS, AND CYTOGENETIC EFFECTS

One frequently expressed concern about RFR is that it may be mutagenic or cause cancer. As suggested by Ames (1979), mutagenesis and carcinogenesis are believed to be related, and indeed many chemicals are screened for potential carcinogenicity with bacterial mutation tests.

Several studies for mutagenic effects of RFR at various frequencies, power densities, and durations have been done on bacteria and yeasts by Blackman et al. (1976), Dutta et al. (1979), and Dardalhon et al. (1981). No mutagenic effects attributable to RFR exposure were reported.

Four studies for mutagenic effects of RFR in fruit flies also yielded negative results. In the first study, Pay et al. (1972) exposed male flies to 2.45-GHz RFR at 6,500, 5,900, and 4,600 mW/cm^2 for 45 min. The test consisted of mating the exposed males to females to determine RFR effects on fertility and then remating the offspring to determine the presence of recessive lethal mutations. The results were negative in both cases. In the second study, Mickey et al. (1975) exposed flies to pulsed RFR at 20-35 MHz at an unstated power density for 4 h. The test consisted of observing for nondisjunction of X and Y chromosomes at mating, and the results again were negative. In the third study, Dardalhon et al. (1977) exposed flies to 17- and 73-GHz RFR at 60 to 100 mW/cm^2 for 2 h. No mutations were found. Last, Hamnerius et al. (1979) exposed fly embryos to 2.45-GHz RFR for 6 h at an SAR of 100 W/kg, corresponding to about 200 mW/cm^2. The test system was designed to measure the frequency of somatic mutations for eye pigmentation. As a positive control, flies were exposed to X-rays, a known mutagenic agent. No mutations were found in the specimens exposed to the RFR.

Several studies have been conducted for possible cytogenetic effects of exposure to RFR. Such studies usually involve two types of observations: (1) abnormalities in chromosomes such as fragmentation, fusion, and interchromosomal bridges at the metaphase stage of mitosis; and (2) sister chromatid exchanges.

Chen et al. (1974) exposed Chinese hamster cells and human amnion cells in vitro to 2.45-GHz RFR at power densities ranging from 200 to 500 mW/cm^2 for durations ranging from 1.5 to 20 min. Various chromosome aberrations were observed; but the incidence of aberrations did not increase with increasing power density or exposure duration, and the incidence in exposed cells was not significantly different from that in control cells.

Stodolnik-Baranska (1974) exposed human lymphocyte cultures to 2.95-GHz pulsed RF (pulse characteristics not given) at 20 or 7 mW/cm^2 average power density for periods ranging from 10 min to 4 h. Exposure at 20 mW/cm^2 for 10 min or longer produced chromosome aberrations, but none were reported for exposure at 7 mW/cm^2 for 4 h. The author noted the occurrence of a "slight" temperature increase in cultures exposed at 20 mW/cm^2 but none in cultures exposed at 7 mW/cm^2. The results suggest that if RFR does cause an increase in chromosome abnormalities, the effect may have a power density threshold.

Two studies reported effects of RF on sister chromatid exchange. In the first study, Livingston et al. (1977) exposed Chinese hamster ovary cells in vitro to 2.45-GHz RFR at unstated power density levels and durations. Sister chromatid exchanges were observed in RFR-exposed cells; however, the same level of exchanges was produced in control cells by heating them to the same temperature as that produced by RFR exposure. The authors concluded that the production of sister chromatid exchanges is not related to RFR exposures per se. In the second study, McRee et al. (1981) exposed mice to 2.45-GHz RFR at 20 mW/cm^2, corresponding to an SAR of 21 W/kg, 8 h per day for 28 days. Incidences of sister chromatid exchange in bone marrow cells of exposed mice, sham-exposed control mice, and standard control mice were compared. No statistically significant differences were detected.

A study by Prausnitz and Susskind (1962) implied an association between RFR exposure and cancer incidence. They exposed male mice to pulsed 9.27-GHz RFR at 100 mW/cm^2 average power density for 4.5 min per day, 5 days per week, for 59 weeks. Each day's exposure was equal to one-half of the acute LD-50 of the animals. The major results were (1) progressive testicular atrophy; (2) death of some mice during exposure; (3) a 65% survival rate for the exposed mice at the end of the exposure series as compared with only 50% for the control; (4) the presence of liver abscesses in both groups at necropsy; and (5) leukosis among both groups, with a higher incidence for the exposed mice. Leukosis was described in the paper as "cancer of the white blood cells."

The authors attributed deaths in both groups to a pneumonia infection accidentally introduced into the colony during the experiment, and suggested that the higher survival rate of the exposed mice was due to the protective effect of the "fever" induced by the daily exposures. However, the authors apparently confused leukosis with leukemia or cancer of the circulatory system. Leukosis is defined as an abnormal rise in the number of circulating white blood cells, which can arise from various causes, including stress, endocrine disturbances, and infection such as that causing the liver abscesses found.

In addition, two other factors must be considered. First, the incidence of leukosis was greater in the exposed mice, but their survival was also greater. This would be considered unusual for most forms of mouse leukemia. Second, in the exposed mice the incidence of leukosis was greater during but not following exposure. This would imply that spontaneous remission of the "cancer" occurred after cessation of exposure. For true cancer, this would be considered improbable. Overall, the data did not provide any valid evidence that chronic RFR exposure induced any form of cancer in the exposed mice.

In another study involving chronic exposure, Spalding et al. (1971) exposed mice to 800-MHz RFR at 43 mW/cm^2 for 2 h per day, 5 days per week, for 35 weeks. Some deaths occurred during exposure and were attributed to thermal effects caused by faulty positioning of the animal holders. The mean life span of the remaining exposed mice did not differ significantly from that of the controls, general indications of health were the same in the two groups, and the incidence of cancer was the same in the exposed and control mice.

Baum et al. (1976) exposed rats to EMP (electromagnetic pulses) at a rate of 5 per second continuously for 94 weeks. The spectrum of the EMP corresponded to an RFR center frequency of 450 MHz, and each pulse had an intensity of 447 kV/m. The exposures had no effect on blood chemistry, blood count, bone marrow cellularity, fertility, embryo development, cytology, histology, or cancer incidence.

In the first of two studies, Varma and Traboulay (1976) sought to induce dominant lethal mutations by exposing the testes of mice to 1.7 GHz at 50 mW/cm^2 for 30 min or at 10 mW/cm^2 for 80 min. In the second study, Varma et al. (1976) exposed the testes of mice once to 2.45-GHz RFR at 100 mW/cm^2 for 10 min; 3 times in 1 day for 10 min each at 50 mW/cm^2; or 4 times in 2 weeks for 10 min each time at 50 mW/cm^2. In each study, the test consisted of breeding the exposed males to separate groups of unexposed females once each week for 7 to 8 weeks after exposure. Females were killed on the 12th day of gestation, and the uteri were scored for number of implants and number of resorption sites. The authors concluded from the first study that 1.7-GHz RFR was mutagenic under both conditions of exposure, and from the second study that 2.45-GHz RFR was mutagenic for the single 10-min exposure at 100 mW/cm^2 but not for the multiple exposures over time at 50 mW/cm^2.

These studies had a number of flaws. In the first study, errors made in tabulating the data led to uncertainty about the reliability of the numbers presented. In the second study, the fetal late-mortality rates were significantly higher for the exposed mice than for the controls, raising the question of what factors other than RFR might be causing dominant lethal effects. In both studies, systematic errors were made in the computation of the chi-square statistic used to evaluate the significance of the supposed mutagenic effect. If the chi-square is correctly computed, the first study would show a marginal but significant increase in the number of lethal mutations for the study as a whole but not for individual weeks of the study, and the second study would show no increase at all.

In addition, the incidence of dominant lethal mutations in control animals differed significantly for the two studies (1% in the first, 5% in the second), leading to questions about the quality of the animal source and the

reliability of the scoring. If the control results from both studies were combined, no mutagenic effect would be evident at any frequency, power density, or duration. Finally, the exposed mice were anesthetized, a condition under which their thermoregulation was impaired, so the temperature increases in the testes may have been much higher than would be predicted from the exposure parameters.

In another study for dominant lethal mutations, Berman et al. (1980) exposed unanesthetized rats to 2.45-GHz RFR at power densities ranging from 5 to 28 mW/cm^2 for 3 h daily, 5 days a week, for up to 3 months. No increases in dominant lethal mutations were evident. Temporary sterility, as indexed by fewer pregnancies, was seen at 28 mW/cm^2 but not at lower power densities. At 28 mW/cm^2, there were significant increases in rectal and intratesticular temperatures.

Two studies searched for possible effects of RFR on mechanisms involved in repair of cellular DNA. Meltz and Walker (1981) examined whether there were any alterations in DNA repair induced by 350-MHz or 1.2-GHz RFR in normal human fibroblasts maintained in vitro after the DNA was damaged by a selected dose of ultraviolet light. Power densities of 1 or 10 mW/cm^2 caused no perturbation of the DNA repair process. Brown et al. (1981) treated mice with streptozocin, a mutagenic/carcinogenic agent known to damage DNA in the rodent liver, and exposed the mice to 400-MHz RFR to determine if excision repair of the DNA would be inhibited. They found that power densities of 1.6 and 16 mW/cm^2, corresponding to SARs of 0.29 and 2.9 W/kg, did not alter the level of excision repair.

In summary, there is no evidence that exposure to RFR induces mutations in bacteria, yeasts, or fruit flies. The results of two studies indicated that RFR induces mutations in mammals; critical review has cast doubt on these findings. Other studies have shown no mutagenic effects of RFR on mammals; evidence for cytogenetic effects is mixed. The lowest power density at which such effects were reported was 20 mW/cm^2, in Stodolnik-Baranska (1974); however, Chen et al. (1974) failed to find cytogenetic effects at 200-500 mW/cm^2. Last, there is no credible evidence that chronic exposure to RFR induces any form of cancer in animals, even at power densities as high as 100 mW/cm^2.

TERATOGENESIS AND DEVELOPMENTAL ABNORMALITIES

Teratogenesis in mammals is the production of physical defects in conceptuses that affect their in-utero development. The term "developmental abnormalities" as used here refers to processes affecting the development of infants after birth. Teratogenic and developmental abnormalities occur naturally at a low rate in most animal species, and relatively little is known about their cause. In a few cases, however, specific agents have been shown to cause significant teratogenic effects; hence, the possibility of teratogenic effects from RFR is an appropriate matter of public concern.

Several studies of RFR teratogenesis in Tenebrio molitor, the darkling beetle, notably those of Carpenter and Livstone (1971), Lindauer et al. (1974), Liu et al. (1975), and Green et al. (1979), indicated that relatively low levels of RFR would produce developmental abnormalities in Tenebrio pupae. In a follow-up study, however, Pickard and Olsen (1979) reported that the number of developmental anomalies depend on such factors as the source of the larvae and the diet fed to them before they entered the pupal stage. This

study also reported that production of developmental anomalies under worst conditions required exposure for 2 h at a mean SAR of 54 W/kg, which was equivalent to about 192 mW/cm^2.

McRee and Hamrick (1977) exposed arrays of Japanese-quail eggs to 2.45-GHz CW RFR at 5 mW/cm^2, corresponding to an SAR of about 4 W/kg, for 24 h per day during the first 12 days of development. They found no gross deformities in quail killed and examined 24 to 36 h after hatching, and no significant differences in total body weight between RFR- and sham-exposed groups or the weights of the heart, liver, gizzard, adrenals, and pancreas. Blood tests showed statistically significant higher hemoglobin and lower monocyte counts in the RFR-exposed birds, but no differences in the other blood parameters. The differences in mean temperature from egg to egg in the RFR-exposed arrays were as much as 0.5°C, rendering it difficult to associate these positive findings with RFR per se.

In another study, Hamrick et al. (1977) reared the birds from similarly exposed arrays of eggs for 5 weeks after hatching. No significant differences in mortality or mean body weights at 4 and 5 weeks were found between RFR- and sham-exposed groups.

Teratogenic effects of RFR have been reported in several studies with mice and rats. Rugh et al. (1974, 1975) exposed pregnant mice on gestation day 8 to 2.45-GHz RFR at 123 mW/cm^2 for 2 to 5 min, corresponding to doses in the range 3-8 cal/g. On gestation day 18, the litters were examined for resorptions and for dead, stunted, malformed, and apparently normal fetuses. No abnormalities were reported at doses less than 3 cal/g, which corresponded to about 25% to 30% of the lethal dose for these animals. At doses above 3 cal/g, some abnormalities were obtained, notably exencephaly or brain hernia.

Berman et al. (1978) exposed pregnant mice to 2.450-GHz RFR for 100 min daily on gestation days 1 through 17 at 3.4-14.0 mW/cm^2, or on gestation days 6 through 15 at 28 mW/cm^2. Control mice were sham exposed similarly. All mice were euthanized on day 18, and their uteri were examined for the number of resorbed and dead conceptuses and live fetuses. The live fetuses were examined for gross structural alterations and weighed. Ten types of anomalies were tabulated by the numbers of litters affected. Of 318 RFR-exposed litters, irrespective of power density, 27 (8.5%) had one or more live abnormal fetuses versus 12 of 336 (3.6%) of the sham-exposed litters. For most of the specific anomalies, the numbers of litters affected were either too small for statistical treatment or no RFR-related pattern was apparent.

The mean live fetal weights of the litters exposed at power densities of 14 mW/cm^2 or lower were not significantly different from those of the corresponding sham-exposed litters. By contrast, for mice exposed at 28 mW/cm^2 and permitted to come to term, the mean weight of their offspring at 7 days of age was about 10% less than that of control mice. However, there were no differences in survival rate between RFR-exposed and control offspring.

In a subsequent investigation, Berman et al. (1982) exposed a group of pregnant mice to 2.45-GHz RFR at 28 mW/cm^2 for 100 min daily on gestation days 6 through 17. Another group was similarly sham exposed. The mice in half of each group were examined on gestation day 18. The incidence of pregnancy; the numbers of live, dead, and resorbed fetuses; and the total number of fetuses were similar for the exposed and sham-exposed mice. However, the mean body

weight of the live fetuses in the RFR group was 10% smaller than that of the sham-exposed group, a finding consonant with the investigation's previous results. In addition, ossification of sternal centers was significantly delayed in the RFR mice.

The mice in the other half of each group were permitted to come to term. At 7 days of age, the mean body weight of the suckling mice of the RFR group also was 10% smaller than for the sham group. As before, the survival rate was not affected, but the growth retardation was permanent.

In the first of two regimens, Chernovetz et al. (1975) exposed one group of pregnant mice to 2.45-GHz RFR for 210 min on gestation day 11, and one each of three other groups on days 12, 13, and 14; totaling 20. The estimated mean SAR was 38 W/kg and the total energy absorbed was 5.44 cal/g, which was just sublethal. Four other groups were similarly sham exposed. In addition, eight groups were injected with cortisone, known to be teratogenic. Four of these were similarly exposed to RFR and the other four were sham exposed. All mice were euthanized on day 19, the numbers of implantations and resorptions were counted, and the fetuses were examined for structural abnormalities.

There were no statistically significant differences in the percentage of fetal mortality or structural abnormalities between the RFR and sham groups not administered cortisone, and no dependence on gestation day of treatment. However, the percentage of normal fetuses was 61% for those injected with cortisone and sham exposed, and 50% for the cortisone-with-RFR groups. These percentages were significantly lower than those for the noncortisone group (both 81%), but they did not differ significantly from each other.

In the second regimen used by Chernovetz et al. (1975), the treatments were similar, but the exposures were done only on gestation day 14 and involved a total of 60 dams equally divided among the four treatments. All dams carried to term, and the numbers of pups that survived to weaning at postpartum age 21 days were noted. The noncortisone RFR group produced 93 pups vs 81 for the noncortisone sham group, but the difference was not significant. However, the cortisone RFR group yielded 25 pups and the cortisone sham group only 2. These values were significantly lower than those for the noncortisone groups, and the difference between them was significant. It is tempting to suggest that the higher survival rate of the cortisone RFR group relative to the cortisone sham group indicates that exposure to RFR may afford some protection against teratogenic agents such as cortisone.

In general, these results indicate that absorption of about 5 cal/g of 2.45-GHz RFR is not teratogenic to mice, a finding that is at variance with those of Rugh et al. (1974, 1975) and Berman et al. (1978). Among the possible reasons for these apparently contradictory findings are differences in exposure systems, use of multiple vs individual animal exposures, gross uncertainties in actual doses, mouse-strain difference, variations in dam handling, and differences in gestation day of treatment.

Several similar studies were conducted with pregnant rats. Chernovetz et al. (1977) sham exposed or exposed pregnant rats to 2.45-GHz RFR at a mean SAR of 31 W/kg for 20 min on only one day during gestation days 10 through 17. At this SAR, the colonic temperature increased by 3.5°C. They also exposed rats to infrared radiation (IR) at a temperature that produced the same

colonic temperature rise as the RFR. Of 64 rats studied, 7 dams died after RFR exposure, 3 died after IR exposure, and none of the sham-exposed rats died.

On gestation day 19, the 54 surviving dams were euthanized and the numbers of implantations and resorptions were counted. Also, each fetus was examined for morphological abnormalities and its viability and mass were determined. The percentages of living fetuses per dam were about 98% each for the sham and IR groups but only 87% for the RFR group, a statistically significant decrease. The mean fetal mass for the shams was 1.63 g, and the values for the IR and RFR groups were 1.53 and 1.54 g, respectively, both significantly lower than the mean for the shams. No structural abnormalities were evident in any of the 468 formed fetuses, all of which were alive when taken, but severe edema and hemorrhagic signs were endemic in the IR and RFR groups.

The brains of 60 fetuses from sham-, IR-, and RFR-exposed animals were assayed for norepinephrine (NE) and dopamine (DA). The average level of NE for the RFR group was significantly lower than that for the shams but only marginally lower than that for the IR group. The average levels of DA ranked similarly, but the differences were not statistically significant. In their discussion, the authors concluded that "considered in sum, our findings could be taken as evidence that a brief but highly thermalizing application of 2,450-MHz microwaves or of infrared energy have biological effects both comparable and different when averaged colonic temperature changes are equal."

One problem with this investigation was the small number of rats used (a point recognized by the investigators), which necessitated averaging the data in each group over the 10- to 16-day gestation period. This questionable procedure, both biologically and statistically, made it difficult to assess the validity of either the positive or negative results of this investigation.

Berman et al. (1981) exposed 70 rats to 2.45-GHz CW RFR for 100 min daily on gestation days 6 through 15 at 28 mW/cm^2 for an estimated SAR of 4.2 W/kg. The mean colonic temperature at the end of each exposure was 40.3°C. A group of 67 rats was similarly sham exposed. No significant differences between groups were found in pregnancy rates; numbers of live, dead, or total fetuses; incidences of external, visceral, or skeletal anomalies or variations; or body weight of live fetuses.

The investigators surmised "that this lack of an effect may hold true at any exposure level less than that which will kill a significant number of the dams by hyperthermia." They also concluded that the rat is an inappropriate model for determining whether RFR would be teratogenic to humans in exposure situations not lethal for the dams. They then suggested that the mouse fetus is a more appropriate model for assessing such human risk. However, this point is open to question, especially for studies involving chronic low-level exposures. Most of the recent results with mice indicate the existence of a threshold SAR (or power density) for teratogenesis, and the effects above the threshold were evidently due to the heat produced by the RFR. Because the thermoregulatory systems of both the rat and mouse are much less efficient than the human system, neither kind of rodent appears to be a satisfactory model for studying RFR teratogenesis. Any of the nonhuman primates would be more suitable.

In a study designed primarily for seeking possible effects of chronic RFR exposure on mother-offspring behavioral patterns and the EEG, Kaplan et al. (1982) exposed 33 female squirrel monkeys near the beginning of the second trimester of pregnancy to 2.45-GHz RFR at whole-body SARs of 0.034, 0.34, or 3.4 W/kg for 3 h per day, 5 days per week, until parturition. The 3.4-W/kg SAR was equivalent to about 10 mW/cm^2. Eight pregnant monkeys were sham exposed for the same periods. After parturition, 18 of the RFR-exposed dams and their offspring were exposed to RFR for an additional 6 months; then the offspring were exposed without the dams for another 6 months.

No differences were found between RFR- and sham-exposed dams in the numbers of live births or in the growth rates of the offspring. The major difference between RFR- and sham-exposed offspring was that 4 of the 5 exposed at 3.4 W/kg both prenatally and after birth unexpectedly died before 6 months of age. These mortality values were too small to place much confidence in statistical inferences. Moreover, these results were not confirmed in a follow-up study of mortality per se reported in Kaplan et al. (1982), in which sufficient numbers of squirrel monkeys were used for adequate statistical treatment.

In summary, in the studies showing demonstrable teratogenic effects of exposure to RFR, power densities or SARs were used that were capable of producing significant heat loads in the animals. In general, the results indicate that a threshold of heat induction or core-temperature increase must be exceeded before teratogenic effects are produced and that RFR per se is not teratogenic.

Cont'd

NERVOUS SYSTEM

Several types of studies have been conducted on effects of RFR on the nervous systems of animals. Such studies have been emphasized in the USSR, where RFR is believed to stimulate the nervous system directly and thereby cause a variety of physiological effects. Many U.S. scientists tend to doubt that RFR interacts directly with the nervous system except, possibly, under special circumstances; they consider most effects of RFR on the nervous system to be indirect results of other physiological interactions.

RFR Hearing Effect

Cont'd

Humans in the vicinity of some types of pulsed radar systems have perceived individual pulses of RFR as audible clicks (without the use of any electronic receptors). This phenomenon has attracted much interest, especially in the United States, because it has often been cited as evidence that nonthermal effects can occur and because one hypothesized mechanism for perception was direct stimulation of the central nervous system by RFR.

Various theoretical and experimental studies with both human volunteers and laboratory animals have been conducted to determine the conditions under which pulsed RFR is audible and to investigate the interaction mechanisms involved. Many of the results support the hypothesis that an RFR pulse having the requisite pulse power density and duration can produce a transient thermal gradient large enough to generate an elastic shock wave at some boundary between regions of dissimilar dielectric properties in the head, and that this shock wave is transmitted to the middle ear where it is perceived as a click.

Persons with impaired hearing are unable to hear such clicks, and experimental animals in which the cochlea has been destroyed do not exhibit brainstem-evoked responses.

The original demonstration that acoustic transients can be generated in liquids such as water by transient surface-heating with RFR pulses was provided by White (1963). Foster and Finch (1974) extended this work to show that peak audiofrequency pressures generated in water by specific combinations of RFR pulse power density and pulse width would be sufficient for humans to perceive such pulses as auditory clicks. Lin (1977a, 1977b, 1978, 1980) reported detailed theoretical and experimental studies of the RFR auditory effect. His results indicated that the audiofrequencies produced are not dependent on the RFR carrier frequency but on head size.

Cain and Rissman (1976, 1978) used 3.0-GHz RFR to study the auditory effect in two cats, two chinchillas, one beagle, and eight human volunteers. For the animals, surface or brainstem-implanted electrodes were used to measure the responses to RFR pulses and the responses evoked by audio clicks from a speaker. They found that perception of 10-μs pulses required pulse power densities of at least 1.3 W/cm^2 for both cats, 1 and 2 W/cm^2 for the two chinchillas, and 300 mW/cm^2 for the beagle. The eight humans were given standard audiograms. Because such audiograms do not test hearing above 8 kHz, binaural hearing thresholds were also determined for seven of the subjects for frequencies in the range from 1 to 20 kHz. Five of the subjects could detect 15-μs pulses as clicks; the other three required a pulse duration of 200 μs for perception. No correlation between the results and the audiograms was apparent; however, there was a strong correlation between RFR perception and hearing ability above 8 kHz as determined from the binaural thresholds. The average threshold pulse power density for 15-μs pulses was about 700 mW/cm^2; however, three of the subjects were able to perceive 15-μs pulses at a pulse power density of 300 mW/cm^2, a value taken herein as the nominal threshold for humans.

Olsen and Hammer (1980, 1981) and Olsen and Lin (1981) exposed muscle-equivalent and brain-equivalent models to 5.7- and 1.1-GHz RFR, respectively, at high pulse power densities and detected RFR-induced acoustical responses with hydrophone transducers implanted in the models. Lin and Olsen (1981) also reported that they could detect RFR-induced acoustic pressure waves in the brains of anesthetized cats and guinea pigs by means of a piezoelectric transducer implanted in the cortex.

In conclusion, the preponderance of experimental results indicates that auditory perception of RFR pulses is due to induction of thermoelastic waves in the head rather than to direct brain stimulation by the RFR. Also, because individual pulses can be perceived, it is not meaningful to calculate average power densities for two or more widely spaced pulses and cite such values as evidence that the phenomenon is nonthermal in nature. Almost all of the results are consistent with the thermal expansion theory.

Con'td

Calcium Efflux

Adey (1979, 1980, 1981a, 1981b) and Blackman et al. (1979, 1980a, 1980b) have reported that exposure of brain-tissue samples from newly hatched chicks to 50-, 147-, or 450-MHz RFR that is amplitude-modulated with frequencies in a narrow band around 16 Hz altered the rate of exchange of calcium ions between

the tissue and its bathing fluid. They also indicated that the effect was absent for unmodulated RFR but occurred for exposure to 16-Hz modulation alone. For the modulated RFR, the incident average power densities effective in altering the rate of calcium exchange lie between approximately 0.1 and 3.6 mW/cm^2. Within this range, however, not all power densities are effective. There appear to be narrow, effective power-density "windows." Calculations of internal field intensity by Joines and Blackman (1980, 1981) indicate that this factor is important in predicting effectiveness. The mechanisms whereby modulation effects are mediated are speculative.

Of additional interest is a report by Albert et al. (1980) that 16-Hz amplitude-modulated 147-MHz RFR at 2.0 mW/cm^2 increases calcium efflux from pancreatic tissue slices to approximately the same extent as that from neonate chick-brain tissue incubated and exposed under similar conditions. An attempt by Shelton and Merritt (1981) to obtain alterations in calcium efflux from rat brain tissue by use of pulse-modulated 1-GHz RFR was unsuccessful. It is uncertain whether these negative findings were a result of differences in brain tissue, exposure parameters, carrier frequency, or type of modulation.

All of the above studies were carried out on isolated tissues maintained in physiological solutions. Adey et al. (1982), however, have reported that similar alterations in calcium ion exchange occur for exposed brains of paralyzed live cats irradiated at 3 mW/cm^2 with 450-MHz RFR sinusoidally amplitude modulated at 16 Hz.

The effect is scientifically interesting in that it represents a rare instance where RFR may be producing a biological effect by processes other than thermal mechanisms. Interpreting these results with regard to human health and safety is difficult. First, the phenomenon is subtle; large numbers of samples have to be processed to show a statistically significant effect. Second, the observations are highly variable and difficult to reproduce. Third, the circumstances of the experimental methodology are such that the observations of changes of calcium exchange appear to apply to the surface region of the brain rather than to the brain as a whole. Finally, the phenomenon depends on the amplitude modulation of the RFR in a narrow frequency band around 16 Hz and occurs only in narrow power-density windows within 0.1 and 3.6 mW/cm^2.

Cont'd

Blood-Brain-Barrier Effects

In most organs and tissues of the body, molecules in the blood can freely diffuse into the tissue around the capillaries. However, presumably to protect the brain from invasion by various blood-borne microorganisms and toxic substances, large molecules such as proteins or polypeptides exhibit little or no movement from the blood into the surrounding brain tissue in most regions of the brain. The exact manner by which the movement is prevented is still conjectural, but the process is referred to as the blood-brain barrier, or BBB. The BBB can be "opened" by certain agents, such as ionizing radiation, heat, or chemical substances. Studies have been conducted to examine whether RFR also can alter the BBB permeability of animals to various large molecules.

Sutton et al. (1973), Sutton and Carroll (1979), and Lin (1980, 1982) have reported gross permeability increases in the rat BBB when the brain temperature was raised several degrees by RFR heating or the local SAR was several hundred watts per kilogram. Albert et al. (1977, 1979) and Albert and Kerns (1981) found scattered regions of permeability changes in the brains of Chinese hamsters exposed to 2.45-GHz RFR for 2 h at 20 mW/cm^2. Twenty percent of the sham-exposed animals also showed such changes, which were reversible. Presumably, significant heating of local regions of the brain occurred.

Frey et al. (1975) reported alterations in BBB permeability to fluorescein by use of pulsed RFR at average power densities as low as 0.2 mW/cm^2. These findings could not be repeated by Merritt et al. (1978) or Spackman et al. (1978, 1979).

Oscar and Hawkins (1977) reported increased BBB permeability to radiotracer-labeled molecules at average power densities less than 3 mW/cm^2, with pulsed RFR more effective than CW RFR. Merritt et al. (1978), Preston et al. (1979), and Chang et al. (1982) were unable to confirm these findings. Subsequently Oscar et al. (1981) showed with different techniques that their original findings could be explained as due to increases in local cerebral blood flow rather than as increases in BBB permeability.

In summary, RFR can alter BBB permeability at exposure levels sufficient to cause heating of the brain. Exposure at levels of the order of 1 mW/cm^2, considered insufficient to cause heating, have also been reported to alter BBB permeability; but such results have not been confirmed despite several independent attempts to do so. In one case the original findings may have arisen as a consequence of the experimental techniques used.

Orig Histopathology and Histochemistry of the Central Nervous System Cont'd

Histopathology is defined as the study of diseased or damaged tissues, and histochemistry as the study of the chemical composition of various tissues. Studies of histopathological effects of RFR on the brain have been conducted in both the United States and the USSR. Studies in the USSR have covered a wide range of frequencies, but the dosimetry and methods were inadequately reported for critical review in many instances. Tolgskaya and Gordon (1973) exposed animals (predominantly rats) to RFR between 500 MHz and 1 GHz at 10 mW/cm^2 for 1 h/day for 10 months. They reported detecting various changes from the normal appearance of nerve cells of the brain by unspecified delicate elective neurohistological methods, and that the power density did not raise body temperature. Current knowledge, however, indicates that the method of exposing the animals was such that the SAR must have varied considerably among the animals. The reported changes in appearance were similar to those found by them at 20-240 mW/cm^2 in acute experiments of a frankly thermal nature, and the reported effects in the chronic exposure experiments were probably also of thermal origin.

In the United States, Albert and DeSantis (1975) sought histopathological effects in brains of hamsters exposed to 2.45- GHz RFR at power densities between 10 and 50 mW/cm^2 for periods between 30 min and 24 h and for 22 days. In this study pathological changes were found only in the hypothalamus and subthalamus. Comments after oral presentation of this study noted that the

nature of RFR absorption inside the skull of such a small animal at the frequency used could lead to regions in the brain where the SAR would be tens of times higher than that expected from the nominal power density and that rectal temperature measurements in the animals would not reflect such a condition. The observed pathological effects seem likely to have resulted from thermal processes.

Quantitative studies by Albert et al. (1981a) showed that exposure of rats at 10-46 mW/cm^2 prenatally and postnatally decreased the number of Purkinje cells of the cerebellum significantly. A similar study by Albert et al. (1981b) using the squirrel monkey, however, did not show such an effect. Size differences between the heads and brains of the rat and squirrel monkey may have resulted in high local SAR in regions of the rat brain but not in similar regions of the squirrel monkey brain, again indicating that the observed effects seem likely to have resulted from thermal processes.

One study that examined effects of RFR on brain neurochemistry, by Merritt and Frazer (1975), showed no effects on specific neurotransmitters of mouse brain at 19 MHz for 10-min exposures at an E-field of 6 kV/m or an H-field of 41 A/m. Another study, by Sanders et al. (1980), showed a sequence of 5% to 10% changes of biochemical activity in subcellular components associated with tissue respiration at exposure levels of 5 and 13.8 mW/cm^2. The significance of these latter findings is unclear, but they are unlikely to indicate a hazard because of the wide range of tissue respiration values possible under various environmental and activity situations.

In summary, RFR can cause observable histopathological changes in the central nervous system of animals at power densities of about 10 mW/cm^2 or higher, but these changes appear to be thermal in nature. Under special conditions of frequency and skull size, a focusing effect can be obtained in small rodents, causing local SARs tens of times higher than would normally be expected from whole-body SAR measurements. Such conditions do not occur for the adult human skull. One study has reported small changes in brain-tissue respiratory chain function at a power density of 5 mW/cm^2. Such effects are unlikely to be hazardous to humans at power densities of 1 mW/cm^2 or less.

Cont'd

EEG Studies.

Studies have been conducted to ascertain the effects of RFR on the EEG or other related electrophysiological properties of the CNS. For EEG measurements made after RFR exposure, the time consumed in placing and attaching the electrodes and the variability of placement introduce problems of interpretation. Additionally, if the effects are transient, they may stop when exposure ceases. For studies attempting to measure EEG changes during RFR, the electrodes and leads used to pick up EEG signals also pick up electrical signals directly from the fields, causing artifacts that render the recordings difficult to interpret. Also, indwelling or chronically attached electrodes will perturb the electric fields in their vicinity and produce great enhancement of energy absorption, as noted by Johnson and Guy (1972) and the National Academy of Sciences (NAS, 1979), thereby creating still another artifact in the biological data. To meet these problems, especially designed indwelling electrodes of high-resistivity materials that do not cause field perturbation have been constructed and used in a few of the more recent studies by Tyazhelov et al. (1977), Chou and Guy (1979), and Chou et al. (1982).

Dumanskii and Shandala (1974) and Takashima et al. (1979) used implanted metallic electrodes and reported changes in EEG patterns after acute or chronic exposure of rabbits to RFR. Chou et al. (1982) used implanted electrodes made of carbon instead of metal in an attempt to avoid the field distortion artifact; they reported no significant differences in EEG between rabbits exposed 2 h/day for 3 months at 1.5 mW/cm^2 and control rabbits. Kaplan et al. (1982) used electrodes externally placed after exposure rather than indwelling ones and reported no differences in EEG patterns between control and RFR-exposed squirrel monkeys after more than 12 months of exposure. Rosenstein (1976) exposed rats to RFR from before birth to age 92 days without indwelling electrodes and saw no differences between exposed and control animals when both groups were tested at 140 days of age. Lastly, Chou and Guy (1979) examined EEGs of rabbits with indwelling high-resistance carbon-loaded Teflon electrodes before and during exposure to 2.45-GHz RFR at 100 mW/cm^2. The SAR at the electrodes was about 25 W/kg. No obvious differences were found.

In summary, indwelling metallic electrodes used in studies of the effects of RFR on the EEG or on evoked potentials of the central nervous system may introduce artifactual effects in the preparations under study as well as in the recordings themselves. These artifacts may be minimized by use of electrodes appropriately designed from high-resistivity materials. Experiments in which such specially constructed electrodes were used, or in which electrodes were applied after exposure, showed no evidence of statistically significant differences in EEGs or evoked responses between RFR-exposed and control animals.

UNRESOLVED ISSUES

In this paper, we have critically reviewed selected reports in several RFR-bioeffects topics considered important with regard to possible hazards to humans. Based on the studies examined here and others (USAFSAM-TR-83-1, March 1983) we were not able to cover, we believe that no reliable evidence indicates that chronic exposure to RFR at incident average power densities below 1 mW/cm^2 or at SARs below 0.4 W/kg are hazardous to human health. However, there are several important uncertainties:

(1) Existing epidemiologic studies, though extensive and reasonably well done, are subject to inherent defects such as imprecise classification of the individuals with regard to RFR exposure and unavailability of complete sets of medical reports, death certificates, or health questionnaires.

(2) The most directly applicable experimental evidence relative to possible bioeffects of exposure to the RFR from any specific RFR-emitting system would be from studies in which humans were exposed to the frequencies and waveform characteristics of that kind of system for appropriate durations at the maximum average power densities likely to be encountered. Further quantitative evaluation of many biological endpoints would be necessary. Because such information does not exist, data are obtained from laboratory animals, such as small rodents, used as surrogates for humans--a standard practice for investigating the effects of other agents. Because of the biological differences among species, however, a basic uncertainty is the degree of validity of this practice--which depends in part on the species used, the nature of the agent and its quantitative aspects, and the biological endpoints studied. In investigations of RFR bioeffects, much progress has been made in quantifying

exposures in terms of whole-body SARs and internal SAR distributions in animal carcasses and in physical and mathematical models of various species (including humans). An overview of such work is provided in another paper presented at this NATO Workshop (see Durney, pp. 5-36).

(3) Most experimental data indicating the existence of threshold power densities for various RFR bioeffects were obtained from exposures for relatively short durations. Although it is difficult to conceive of mechanisms whereby long-term RFR exposures at well below threshold values could result in cumulative effects deleterious to health, very few investigations have involved exposure of animals to low-level RFR over a large fraction of their lifetimes. The results of one such study are covered in another paper at this NATO Workshop (see Krupp, pp. 121-133).

(4) Questions of quantitative and/or qualitative differences in bioeffects induced by pulsed versus CW RFR at equivalent average power densities cannot be resolved fully from current knowledge (i.e., some investigators have found no significant differences, whereas others have). Also, it should be noted that although the permissible average power densities in most current and proposed U.S. safety guidelines are applicable to both pulsed and CW RFR, these guidelines do not include maximum allowable pulse power densities per se.

In the light of these uncertainties, the possibility that new information would reveal a significant hazard to humans from chronic exposure to low levels of RFR cannot be dismissed, but seems unlikely.

REFERENCES

Adey, W. R. "Neurophysiologic Effects of Radiofrequency and Microwave Radiation," Bull. N.Y. Acad. Med., Vol. 55, No. 11, pp. 1079-1093 (1979).

Adey, W. R. "Frequency and Power Windowing in Tissue Interactions with Weak Electromagnetic Fields," Proc. IEEE, Vol. 68, No. 1, pp. 119-125 (1980).

Adey, W. R. "Tissue Interactions with Nonionizing Electromagnetic Fields," Physiol. Rev., Vol. 61, No. 2, pp. 435-514 (1981a).

Adey, W. R. "Ionic Nonequilibrium Phenomena in Tissue Interactions with Electromagnetic Fields," in K. H. Illinger, "Effects of Nonionizing Radiation," American Chem. Scc. Series 157, pp. 271-297 (1981b).

Adey, W. R., S. M. Bawin, and A. F. Lawrence. "Effects of Weak Amplitude-Modulated Microwave Fields on Calcium Efflux from Awake-Cat Cerebral Cortex," Bioelectromagnetics, Vol. 3, No. 3, pp. 295-307 (1982).

Albert, E. N. "Reversibility of the Blood-Brain Barrier," Radio Sci., Vol. 14, No. 6S, pp. 323-327 (1979).

Albert, E. N., and M. DeSantis. "Do Microwaves Alter Nervous System Structure?" Ann. N. Y. Acad. Sci., Vol. 247, pp. 87-108 (1975).

Albert, E. N., and J. M. Kerns. "Reversible Microwave Effects on the Blood-Brain Barrier," Brain Res., Vol. 230, pp. 153-164 (1981).

Albert, E., C. F. Blackman, and F. Slaby. "Calcium-Dependent Secretory Protein Release and Calcium Efflux during RF Irradiation of Rat Pancreatic Tissue Slices," Proc. URSI Int. Symposium on Electromagnetic Waves and Biology, Paris, France, pp. 325-330 (June-July 1980).

Albert, E. N., L. Grau, and J. Kerns. "Morphologic Alterations in Hamster Blood-Brain Barrier after Microwave Irradiation," J. Microwave Power, Vol. 12, No. 1, pp. 43-44 (1977).

Albert, E. N., M. F. Sherif, N. J. Papadopoulos, F. J. Slaby, and J. Monahan. "Effect of Nonionizing Radiation on the Purkinje Cells of the Rat Cerebellum," Bioelectromagnetics, Vol. 2, No. 3, pp. 247-257 (1981a).

Albert, E. N., M. F. Sherif, and N. J. Papadopoulos. "Effect of Nonionizing Radiation on the Purkinje Cells of the Uvula in Squirrel Monkey Cerebellum," Bioelectromagnetics, Vol. 2, No. 3, pp. 241-246 (1981b).

Ames, B. N. "Identifying Environmental Chemicals Causing Mutations and Cancer," Science, Vol. 204, pp. 587-592 (1979).

Appleton, B. Results of Clinical Surveys for Microwave Ocular Effects, U. S. Dept. of Health, Education, and Welfare, Public Health Service report, HEW Publication (FDA) 73-8031, BRH/DBE 73-3 (February 1973).

Baum, S. J., M. E. Ekstrom, W. D. Skidmore, D. E. Wyant, and J. L. Atkinson. "Biological Measurements in Rodents Exposed Continuously Throughout Their Adult Life to Pulsed Electromagnetic Radiation," Health Phys., Vol. 30, pp. 161-166 (1976).

Berman, E., H. B. Carter, and D. House. "Tests of Mutagenesis and Reproduction in Male Rats Exposed to 2450-MHz (CW) Microwaves," Bioelectromagnetics, Vol. 1, No. 2, pp. 65-76 (1980).

Berman, E., H. B. Carter, and D. House. "Observations of Rat Fetuses after Irradiation with 2450-MHz (CW) Microwaves," J. Microwave Power, Vol. 16, No. 1, pp. 9-13 (1981).

Berman, E., H. B. Carter, and D. House. "Reduced Weight in Mice Offspring after In Utero Exposure to 2450-MHz (CW) Microwaves," Bioelectromagnetics, Vol. 3, No. 2, pp. 285-291 (1982).

Berman, E., J. B. Kinn, and H. B. Carter. "Observations of Mouse Fetuses after Irradiation with 2.45 GHz Microwaves," Health Phys., Vol. 35, pp. 791-801 (1978).

Bielski, J., A. Sawinska, and J. Pianowska. "Bioelectrical Brain Activity in Employees Exposed to Various Frequencies of Electromagnetic Fields," Proc. URSI, Int. Symposium on Electromagnetic Waves and Biology, Paris, France, pp. 193-195 (June-July 1980).

Blackman, C. F., S. G. Benane, J. A. Elder, D. E. House, J. A. Lampe, and J. M. Faulk. "Induction of Calcium-Ion Efflux from Brain Tissue by Radiofrequency Radiation: Effect of Sample Number and Modulation Frequency on the Power-Density Window," Bioelectromagnetics, Vol. 1, No. 1, pp. 35-43 (1980a).

Blackman, C. F., S. G. Benane, W. T. Joines, M. A. Hollis, and D. E. House. "Calcium-Ion Efflux from Brain Tissue: Power Density vs. Internal Field-Intensity Dependencies at 50-MHz RF Radiation," Bioelectromagnetics, Vol. 1, No. 3, pp. 277-283 (1980b).

Blackman, C. F., J. A. Elder, C. M. Weil, S. G. Benane, D. C. Eichinger, and D. E. House. "Induction of Calcium-Ion Efflux from Brain Tissue by Radiofrequency Radiation: Effects of Modulation Frequency and Field Strength," Radio Sci., Vol. 14, No. 6S, pp. 93-98 (1979).

Blackman, C. F., M. C. Surles, and S. G. Benane. "The Effect of Microwave Exposure on Bacteria Mutation Induction," in C. C. Johnson and M. Shore (eds.), Biological Effects of Electromagnetic Waves, U.S. Department of Health, Education, and Welfare, HEW Publication (FDA) 77-8010, pp. 406-413 (1976).

Brown, R. F., S. V. Marshall, and C. W. Hughes. "Effects of RFR on Excision-Type DNA Repair In Vivo," in J. C. Mitchell (ed.), USAF Radiofrequency Radiation Bioeffects Research Program--A Review, Aeromed. Rev. 4-81, Report No. SAM-TR-81-30, pp. 184-200 (1981).

Burdeshaw, J. A., and S. Schaffer. "Factors Associated with the Incidence of Congenital Anomalies: A Localized Investigation," Report No. XXIII, May 24, 1973-March 31, 1976, EPA 600/1-77-016 (March 1977).

Cain, C. A., and W. J. Rissman. "Microwave Hearing in Mammals at 3.0 GHz," in C. C. Johnson and M. Shore (eds.), Biological Effects of Electromagnetic Waves, U.S. Department of Health, Education, and Welfare, HEW Publication (FDA) 77-8010, pp. 79-88 (1976).

Cain, C. A., and W. J. Rissman. "Mammalian Auditory Responses to 3.0 GHz Microwave Pulses," IEEE Trans. Biomed. Eng., Vol. 25, No. 3, pp. 288-293 (1978).

Carpenter, R. L., and E. M. Livstone. "Evidence for Nonthermal Effects of Microwave Radiation: Abnormal Development of Irradiated Insect Pupae," IEEE Trans. Microwave Theory & Tech., Vol. 19, No. 2, pp. 173-178 (1971).

Chang, B. K., A. T. Huang, W. T. Joines, and R. S. Kramer. "The Effect of Microwave Radiation (1.0 GHz) on the Blood-Brain Barrier in Dogs," Radio Sci., Vol. 17, No. 5S, pp. 165-168 (1982).

Chen, K. M., A. Samuel, and R. Hoopingarner. "Chromosomal Aberrations of Living Cells Induced by Microwave Radiation," Environ. Lett., Vol. 6, pp. 37-46 (1974).

Chernovetz, M. E., D. R. Justesen, N. W. King, and J. E. Wagner. "Teratology, Survival, and Reversal Learning after Fetal Irradiation of Mice by 2,450 MHz Microwave Energy," J. Microwave Power, Vol. 10, pp. 391-409 (1975).

Chernovetz, M. E., D. R. Justesen, and A. F. Oke. "A Teratological Study of the Rat: Microwave and Infrared Radiations Compared," Radio Sci., Vol. 12, No. 6S, pp. 191-197 (1977).

Chou, C.-K., and A. W. Guy. "Carbon-Loaded Teflon Electrodes for Chronic EEG Recordings in Microwave Research," J. Microwave Power, Vol. 14, No. 4, pp. 399-404 (1979).

Chou, C.-K., A. W. Guy, J. A. McDougall, and L.-F. Han. "Effects of Continuous and Pulsed Chronic Microwave Radiation on Rabbits," Radio Sci., Vol. 17, No. 5S, pp. 185-193 (1982).

Cleary, S. F., and B. S. Pasternack. "Lenticular Changes in Microwave Workers. A Statistical Study," Arch. Environ. Health, Vol. 12, pp. 23-29 (1966).

Cleary, S. F., B. S. Pasternack, and G. W. Beebe. "Cataract Incidence in Radar Workers," Arch. Environ. Health, Vol. 11, pp. 179-182 (1965).

Cohen, B. H., A. M. Lilienfeld, S. Kramer, and L. C. Hyman. "Parental Factors in Down's Syndrome--Results of the Second Baltimore Case-Control Study," in E. G. Hook and I. H. Porter (eds.), Population Genetics Studies in Humans, Academic Press, New York, pp. 301-352 (1977).

Dardalhon, M., D. Averbeck, and A. J. Berteaud. "Studies on Possible Genetic Effects of Microwaves in Procaryotic and Eucaryotic Cells," Radiat. Environ. Biophys., Vol. 20, pp. 37-51 (1981).

Dardalhon, M., A. J. Berteaud, and C. Averbeck. "Microwave Effects in Drosophila melanogaster," Abstracts of 1977 International Symposium on the Biological Effects of Electromagnetic Waves, Airlie, Virginia, p. 25 (1977).

Dumanskii, J. D., and M. G. Shandala. "The Biologic Action and Hygienic Significance of Electromagnetic Fields of Superhigh and Ultrahigh Frequencies in Densely Populated Areas," in P. Czerski et al. (eds.), Biologic Effects and Health Hazards of Microwave Radiation, Polish Medical Publishers, Warsaw, pp. 289-293 (1974).

Dutta, S. K., W. H. Nelson, C. F. Blackman, and D. J. Brusick. "Lack of Microbial Genetic Response to 2.45-GHz CW and 8.5- to 9.6-GHz Pulsed Microwaves," J. Microwave Power, Vol. 14, No. 3, pp. 275-280 (1979).

Foster, K. R., and E. D. Finch. "Microwave Hearing: Evidence for Thermoacoustic Auditory Stimulation of Pulsed Microwaves," Science, Vol. 185, pp. 256-258 (1974).

Frey, A. H., S. R. Feld, and B. Frey. "Neural Function and Behavior: Defining the Relationship," Ann. N. Y. Acad. Sci., Vol. 247, pp. 433-439 (1975).

Green, D. R., F. J. Rosenbaum, and W. F. Pickard. "Intensity of Microwave Irradiation and the Teratogenic Response of Tenebrio molitor," Radio Sci., Vol. 14, No. 6S, pp. 165-171 (1979).

Hamnerius, Y., H. Olofsson, A. Rasmuson, and B. Rasmuson. "A Negative Test for Mutagenic Action of Microwave Radiation in Drosphila melanogaster," Mutation Res., Vol. 68, No. 3, pp. 217-223 (1979).

Hamrick, P. E., D. I. McRee, P. Thaxton, and C. R. Parkhurst. "Humoral Immunity of Japanese Quail Subjected to Microwave Radiation during Embryogeny," Health Phys., Vol. 33, pp. 23-33 (1977).

Johnson, C. C., and A. W. Guy. "Nonionizing Electromagnetic Wave Effects in Biological Materials and System," Proc. IEEE, Vol. 60, No. 6, pp. 692-718 (1972).

Joines, W. T., and C. F. Blackman. "Power Density, Field Intensity, and Carrier Frequency Determinants of RF-Energy-Induced Calcium-Ion Efflux from Brain Tissue," Bioelectromagnetics, Vol. 1, No. 3, pp. 271-275 (1980).

Joines, W. T., and C. F. Blackman. "Equalizing the Electric Field Intensity within Chick Brain Immersed in Buffer Solution at Different Carrier Frequencies," Bioelectromagnetics, Vol. 2, No. 4, pp. 411-413 (1981).

Kalyada, T. V., P. P. Fukolova, and N. N. Goncharova. "Biologic Effects of Radiation in the 30-300 MHz Range," in P. Czerski et al. (eds.), Biologic Effects and Health Hazards on Microwave Radiation, Polish Medical Publishers, Warsaw, pp. 52-57 (1974).

Kaplan, J., P. Polson, C. Rebert, K. Lunan, and M. Gage. "Biological and Behavioral Effects of Pre- and Postnatal Exposure to 2450 MHz Electromagnetic Radiation in the Squirrel Monkey," Radio Sci., Vol. 17, No. 5S, pp. 135-144 (1982).

Klimkova-Deutschova, E. "Neurological Findings in Persons Exposed to Microwaves," in P. Czerski et al. (eds.), Biologic Effects and Health Hazards of Microwave Radiation, Polish Medical Publishers, Warsaw, pp. 268-272 (1974).

Lester, J. R., and D. F. Moore. "Cancer Mortality and Air Force Bases," J. Bioelectricity, Vol. 1, No. 1, pp. 77-82 (1982a).

Lester, J. R., and D. F. Moore. "Cancer Incidence and Electromagnetic Radiation," J. Bioelectricity, Vol. 1, No. 1, pp. 59-76 (1982b).

Lilienfeld, A. M., et al. "Foreign Service Health Status Study: Evaluation of Health Status of Foreign Service and Other Employees from Selected Eastern European Posts," Dept. of Epidemiology, School of Hygiene and Public Health, the Johns Hopkins University, Baltimore (31 July 1978).

Lin, J. C. "Further Studies on the Microwave Auditory Effect," IEEE Trans. Microwave Theory and Tech., Vol. 25, No. 11, pp. 938-943 (1977a).

Lin, J. C. "Theoretical Calculations of Frequencies and Thresholds of Microwave-Induced Auditory Signals," Radio Sci., Vol. 12, No. 6S, pp. 237-242 (1977b).

Lin, J. C. Microwave Auditory Effects and Applications, C C Thomas, Springfield, Illinois (1978).

Lin, J. C. "The Microwave Auditory Phenomenon," Proc. IEEE, Vol. 68, No. 1, pp. 67-73 (1980).

Lin, J. C., and M. F. Lin. "Studies and Microwave and Blood-Brain-Barrier Interaction," Bioelectromagnetics, Vol. 1, No. 3, pp. 313-323 (1980).

Lin, J. C., and M. F. Lin. "Microwave Hyperthermia-Induced Blood-Brain-Barrier Alterations," Radiat. Res., Vol. 89, pp. 77-87 (1982).

Lin, J. C., and R. G. Olsen. "Microwave-Induced Pressure Waves in Brains of Cats and Guinea Pigs," Abstracts of Bioelectromagnetics Symposium, Washington, D.C., p. 25 (August 1981).

Lindauer, G. A., M. Liu, G. W. Skewes, and F. J. Rosenbaum. "Further Experiments Seeking Evidence of Nonthermal Biological Effects of Microwave Radiation," IEEE Trans. Microwave Theory & Tech., Vol. 22, No. 8, pp. 790-793 (1974).

Liu, L. M., F. J. Rosenbaum, and W. F. Pickard. "The Relation of Teratogenesis in Tenebrio Molitor to the Incidence of Low-Level Microwaves," IEEE Trans. Microwave Theory and Tech., Vol. 23, No. 11, pp. 929-931 (1975).

Livingston, G. K., C. C. Johnson, and L. A. Dethlefsen. "Comparative Effects of Water Bath and Microwave-Induced Hyperthermia on Cell Survival and Sister Chromatid Exchange in Chinese Hamster Ovary Cells," Abstracts of 1977 International Symposium on the Biological Effects of Electromagnetic Waves, Airlie, Virginia, p. 106 (1977).

McRee, D. I., and P. E. Hamrick. "Exposure of Japanese Quail Embryos to 2.45 GHz Microwave Radiation during Development," Radiat. Res., Vol. 71, pp. 355-366 (1977).

McRee, D. I., G. MacNichols, and G. K. Livingston. "Incidence of Sister Chromatid Exchange in Bone Marrow Cells of the Mouse Following Microwave Exposure," Radiat. Res., Vol. 85, pp. 340-348 (1981).

Meltz, M. L., and K. A. Walker. "Genetic Effects of Microwave Exposure on Mammalian Cells In Vitro," in J. C. Mitchell (ed.), USAF Radiofrequency Radiation Bioeffects Research Program--A Review, Aeromed. Rev. 4-81, Report No. SAM-TR-81-30, pp. 201-214 (1981).

Merritt, J. H., A. F. Chamness, and S. J. Allen. "Studies on Blood-Brain-Barrier Permeability after Microwave Radiation," Rad. and Environ. Biophys., Vol. 15, pp. 367-377 (1978).

Merritt, J. H., and J. W. Frazer. "Effects of 19-MHz RF Radiation on Neurotransmitters in Mouse Brain," USAF School of Aerospace Medicine, Brooks AFB, Texas, Report SAM-TR-75-28 (August 1975).

Mickey, G. H., J. H. Heller, and E. Snyder. Non-thermal Hazards of Exposure to Radio-Frequency Fields," Final Report, Microwave Studies, Office of Naval Research (July 1975).

NAS (National Academy of Sciences). "Analysis of the Exposure Levels and Potential Biologic Effects of the PAVE PAWS Radar System" (1979).

NTIA. "Microwave Radiation of the U. S. Embassy in Moscow and Its Biological Implications: An Assessment," Report NTIA-SP-81-12, National Telecommunications and Information Administration, Department of Commerce (March 1981).

Olsen, R. G., and W. C. Hammer. "Microwave-Induced Pressure Waves in a Model of Muscle Tissue," Bioelectromagnetics, Vol. 1, No. 1, pp. 45-54 (1980).

Olsen, R. G., and W. C. Hammer. "Evidence for Microwave-Induced Acoustical Resonances in Biological Material," J. Microwave Power, Vol. 16, Nos. 3 & 4, pp. 263-269 (1981).

Olsen, R. G., and J. C. Lin. "Microwave Pulse-Induced Acoustic Resonances in Spherical Head Models," IEEE Trans. Microwave Theory and Tech., Vol. 29, No. 10, pp. 1114-1117 (1981).

Oscar, K. J., S. P. Gruenau, M. T. Folker, and S. I. Rapoport. "Local Cerebral Blood Flow after Microwave Exposure," Brain Res., Vol. 204, pp. 220-225 (1981).

Oscar, K. J., and T. D. Hawkins. "Microwave Alteration of the Blood-Brain-Barrier System of Rats," Brain Res., Vol. 126, pp. 281-293 (1977).

Pay, T. L., E. C. Beyer, and C. F. Reichelderfer. "Microwave Effects on Reproductive Capacity and Genetic Transmission in Drosophila melanogaster," J. Microwave Power, Vol. 7, No. 2, pp. 75-82 (1972).

Pazderova, J. "Workers' State of Health under Long-Term Exposure to Electromagnetic Radiation in the VHF Band (30-300 MHz)," Pracovni Lekarstvi (Czech), Vol. 23, No. 8, pp. 265-271 (October 1971); English Translation: JPRS No. UDC 616-001.228.1-057-07 (1971).

Pazderova, J., J. Pickova, and V. Bryndova. "Blood Proteins in Personnel of Television and Radio Transmitting Stations," in P. Czerski et al. (eds.), Biologic Effects and Health Hazards of Microwave Radiation, Polish Medical Publishers, Warsaw, pp. 281-288 (1974).

Pazderova-Vejlupkova, J. "Influence of Pulsed Microwave Radiation on the Lymphocytes of Rats," Abstracts of Bioelectromagnetics Symposium, Seattle, Washington, p. 477 (1979).

Peacock, P. B., J. W. Simpson, and C. A. Alford. "Congenital Anomalies in Alabama," J. Med. Assoc. Ala., Vol. 41, No. 1, pp. 42-50 (1971).

Pickard, W. F., and R. G. Olsen. "Developmental Effects of Microwaves on Tenebrio; Influences of Culturing Protocol and of Carrier Frequency," Radio Sci., Vol. 14, No. 6S, pp. 181-185 (1979).

Pollack, H. "Epidemiological Data on American Personnel in the Moscow Embassy," Bull. N. Y. Acad. Med., Vol. 55, No. 11, pp. 1182-1186 (1979).

Prausnitz, S., and C. Susskind. "Effects of Chronic Microwave Irradiation on Mice," IRE Trans. Bio-Med. Electron., pp. 104-108 (1962).

Preston, E., E. J. Vavasour, and H. M. Assenheim. "Permeability of the Blood-Brain Barrier to Mannitol in the Rat Following 2450-MHz Microwave Radiation," Brain Res., Vol. 174, pp. 109-117 (1979).

Robinette, C. D., and C. Silverman. "Causes of Death Following Occupational Exposure to Microwave Radiation (RADAR), 1950-1974," in D. G. Hazzard (ed.), Symposium on Biological Effects and Measurement of Radiofrequency/Microwaves, U. S. Department of Health, Education, and Welfare, HEW Publication No. (FDA) 77-8026, pp. 338-344 (1977).

Rodzilsky, B., and J. Olszewski. "Permeability of Cerebral Blood Vessels Studied by Radioactive Iodinated Bovine Albumin," Neurology, Vol. 7, pp. 270-279 (1957).

Rosenstein, L. EEG Investigations in Animals Exposed to Microwave Radiation, Progress Report on Project 2, Topic 4.1, of the U. S.-USSR Cooperative Program, D. McRee (Coordinator), National Institute of Environmental Health Sciences, North Carolina (1976).

Rugh, R., E. I. Ginns, H. S. Ho, and W. M. Leach. "Are Microwaves Teratogenic?" in P. Czerski et al. (eds.), Biological Effects and Health Hazards of Microwave Radiation, Polish Medical Publishers, Warsaw, pp. 98-107 (1974).

Rugh, R., E. I. Ginns, H. S. Ho, and W. M. Leach. "Responses of the Mouse to Microwave Radiation during Estrous Cycle and Pregnancy," Radiat. Res., Vol. 62, pp. 225-241 (1975).

Sadchikova, M. N. "Clinical Manifestations of Reactions to Microwave Irradiation in Various Occupational Groups," in P. Czerski et al. (eds.), Biologic Effects and Health Hazards of Microwave Radiation, Polish Medical Publishers, Warsaw, pp. 261-267 (1974).

Sanders, A. P., D. J. Schaefer, and W. T. Joines. "Microwave Effects on Energy Metabolism of Rat Brain," Bioelectromagnetics, Vol. 1, No. 2, pp. 171-181 (1980).

Shelton, W. W., Jr., and J. H. Merritt. "In Vitro Study of Microwave Effects on Calcium Efflux in Rat Brain Tissue," Bioelectromagnetics, Vol. 2, No. 2, pp. 161-167 (1981).

Siekierzynski, M. "A Study of the Health Status of Microwave Workers," in P. Czerski et al. (eds.), Biologic Effects and Health Hazards of Microwave Radiation, Polish Medical Publishers, Warsaw, pp. 273-280 (1974).

Sigler, A. T., A. M. Lilienfeld, B. H. Cohen, and J. E. Westlake. "Radiation Exposure in Parents of Children with Mongolism (Down's Syndrome)," Bull. Johns Hopkins Hosp., Vol. 117, pp. 374-399 (1965).

Silverman, C. "Epidemiologic Approach to the Study of Microwave Effects," Bull. N. Y. Acad. Med., Vol. 55, No. 11, pp. 1166-1181 (1979).

Spackman, D. H., V. Riley, A. W. Guy, and C.-K. Chou, "Studies of RF Radiation Effects on Blood-Brain-Barrier Permeability Using Fluorescein and Amino Acids," Abstracts of Open Symposium on the Biological Effects of Electromagnetic Waves, Helsinki, Finland, p. 75 (1978).

Spackman, D. H., V. Riley, A. W. Guy, and C. -K. Chou. "Is the Blood-Brain Barrier Altered by RF Irradiation?" Abstracts of Bioelectromagnetics Symposium, Seattle, Washington, p. 362 (1979).

Spalding, J. F., R. W. Freyman, and L. M. Holland. "Effects of 800 MHz Electromagnetic Radiation on Body Weight, Activity, Hematopoiesis, and Life Span in Mice," Health Phys., Vol. 20, pp. 421-424 (1971).

Stodolnik-Baranska, W. "The Effects of Microwave on Human Lymphocyte Cultures," in P. Czerski et al. (eds.), Biologic Effects and Health Hazards of Microwave Radiation, Polish Medical Publishers, Warsaw, pp. 189-195 (1974).

Sutton, C. H., and F. B. Carroll. "Effects of Microwave-Induced Hyperthermia on the Blood-Brain Barrier of the Rat," Radio Sci., Vol. 14, No. 6S, pp. 329-334 (1979).

Sutton, C. H., R. L. Nunnally, and F. B. Carroll. "Protection of the Microwave-Irradiated Brain with Body-Core Hypothermia," Cryobiology, Vol. 10, p. 513 (1973).

Takashima, S., B. Onaral, and H. P. Schwan, "Effects of Modulated RF Energy on the EEG of Mammalian Brains," Radiat. Environ. Biophys., Vol. 16, pp. 15-27 (1979).

Tolgskaya, M. S., and Z. V. Gordon. Pathological Effects of Radio Waves (translated from the original Russian text published by Meditsina Press, Moscow, 1971), Consultants Bureau, New York-London (1973).

Tyazhelov, V. V., R. E. Tigranian, and E. P. Khizhniak. "New Artifact-free Electrodes for Recording of Biological Potentials in Strong Electromagnetic Fields," Radio Sci., Vol. 12, No. 6S, pp. 121-123 (1977)

Tyazhelov, V. V., R. E. Tigranian, E. O. Khizhniak, and I. G. Akoev "Some Peculiarities of Auditory Sensations Evoked by Pulsed Microwave Fields," Radio Sci., Vol. 14, No. 6S, pp. 259-263 (1979).

USAFSAM-TR-81-24, "Review and Analysis of Radiofrequency Radiation Bioeffects Literature," USAF School of Aerospace Medicine, Brooks AFB, Texas (November 1981).

USAFSAM-TR-82-16, "Review and Analysis of Radiofrequency Radiation Literature: Second Report," USAF School of Aerospace Medicine, Brooks AFB, Texas (May 1982).

USAFSAM-TR-83-1, "Bioeffects of Radiofrequency Radiation: A Review Pertinent to Air Force Operations," USAF School of Aerospace Medicine, Brooks AFB, Texas (March 1983).

USAFSAM-TR-84-6, "Review and Analysis of Radiofrequency Radiation Literature: Third Report," USAF School of Aerospace Medicine, Brooks AFB, Texas (March 1984).

USAFSAM-TR-84-17, "Review and Analysis of Radiofrequency Radiation Literature: Fourth Report," USAF School of Aerospace Medicine, Brooks AFB, Texas (May 1984).

Varma, M. M., E. L. Dage, and S. R. Joshi. "Mutagenicity Induced by Non-Ionizing Radiation in Swiss Male Mice," in C. C. Johnson and M. Shore (eds.), Biological Effects of Electromagnetic Waves, U.S. Department of Health, Education, and Welfare, HEW Publication (FDA) 77-8010, pp. 397-405 (1976).

Varma, M. M., and E. A. Traboulay, Jr. "Evaluation of Dominant Lethal Test and DNA Studies in Measuring Mutagenicity Caused by Non-Ionizing Radiation," in C. C. Johnson and M. Shore (eds.), Biological Effects of Electromagnetic Waves, U.S. Department of Health, Education, and Welfare, HEW Publication (FDA) 77-8010, pp. 386-396 (1976).

White, R.M. "Generation of Elastic Waves by Transient Surface Heating," J. Appl. Phys., Vol. 34, pp. 3559-3567 (1963).

Zaret, M., S. Cleary, B. Pasternack, and M. Eisenbud. "Occurrence of Lenticular Imperfections in the Eyes of Microwave Workers and Their Association with Environmental Factors," Rome Air Development Center Report RADN-TN-61-226, New York University (1961).